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The Threshold for Hypoxia Effects on Perceptual-Motor Performance

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The hypoxia threshold for a decrement in perceptual-motor performance was determined with six subjects using a serial choice response time task at two levels of stimulus brightness. Low-oxygen mixtures were used to reduce SaO_2 (arterial oxyhemoglobin saturation) to hypoxic levels ranging from 86% to 76% in steps of 2%. These values correspond to altitudes ranging from 8900 ft to 11 400 feet. Response time was slowed in a dose-dependent manner with a significant effect becoming apparent at an SaO_2 of 82% (10 000 feet). The slope of the dose-response function was steeper for the low than for the high brightness condition. These results provide a threshold estimate of 9750 feet for performance decrements due to hypoxia and point to the disruption of vision as a factor influencing this decrement.

INTRODUCTION

The minimum altitude at which perceptual-motor decrements due to hypoxia can be detected is a controversial issue having implications for flying safety. Tune (1964) suggested an altitude of 10 000 feet as a first approximation but did not preclude the possibility that an effect could be detected at a lower level. In fact, Ernsting (1978, 1984) recommended that cabin altitudes should be maintained at or below 8000 feet in the interests of safety. This suggestion was based primarily on a study by Denison, Ledwith, and Poulton (1966), who reported increased reaction times at 8000 feet using a spatial transformation task (the Mannikin test) with the subjects pedaling a bicycle ergometer at 27 W to simulate pilot workload.

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In contrast, other workers have been unable to obtain consistent performance decrements below 12 000 feet with a variety of tasks other than the Mannikin test (Crow and Kelman, 1971, 1973; Kelman and Crow, 1969; Kelman, Crow, and Bursill, 1969). Recently, Fowler, Paul, Porlier, Elcombe, and Taylor (1985) used the Mannikin test to investigate the possibility that it is particularly sensitive to hypoxia. SaO_2 (arterial oxyhemoglobin saturation) was monitored, and increased reaction times were found at approximately 12 000 feet but not at 8000 feet. These workers proposed that the reason for the effect reported by Denison et al. (1966) was probably a combination of hypoxia, exercise, and hypoventilation that led to an unforseen increase in the effective altitude (defined by SaO_2) over the nominal altitude (defined by the partial pressure of the oxygen in the breathing mixture).

It has been demonstrated that severe hyp-

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oxia slows performance on a serial choice response time (SCRT) task (Fowler, White, Wright, and Ackles, 1982). Part of this slowing was attributed to visual degradation, because response time increased disproportionately when stimulus brightness was lowered. Vision is particularly sensitive to hypoxia (McFarland, 1963), with brightness discrimination showing a definite effect at 8000 feet (Hecht, Hendley, Frank, and Haig, 1946). This raises the possibility that visual factors influence the threshold dose-response function for the effects of hypoxia on perceptual-motor performance.

In summary, present evidence indicates that hypoxia degrades performance at 12 000 feet but not at 8000 feet. The first purpose of this experiment was to arrive at a more precise estimate of the perceptual-motor threshold for hypoxia by constructing a dose-response function using the SCRT. The second purpose was to investigate the influence of visual factors on the dose-response function by manipulating the brightness of the SCRT stimuli.

METHOD

Subjects

Six subjects (3 men and 3 women), aged between 21 and 27 years, volunteered for this experiment and were paid for their participation. Each subject was medically screened and signed an informed-consent document.

Apparatus

The SCRT consisted of a module (61 cm × 61 cm × 10.6 cm) mounted on a stand so that the square face was in the horizontal plane. The upper face of the module was painted flat black and contained five push buttons (Microswitch, Model PK 85022), the heads of which projected 1.5 cm above the surface of the cabinet. Each head had a 2-cm diameter, 20-gauge aluminum unpainted disc cemented to it. A red LED (light emitting diode, Fairchild, Model FLV560) was mounted outside of, and 3.18 cm from the center of, each push button, with respect to a line radiating from the center of the cabinet to the push buttons. The buttons were arranged in the shape of a pentagon at an average distance of 20 cm apart. The brightness of the LEDs could be adjusted to two levels, high and low. Under the experimental lighting conditions (a desk lamp with a 40-watt bulb positioned approximately 1 m to one side and behind the subject), luminance measures taken with a calibrated light meter (Spectra Model UB 1/2 with microspectar lens) indicated 4.88 cd/m^2 with the LED on low setting and 50.0 cd/m^2 on high setting. A PDP 11/04 computer was used to control a sequence of events in which an LED was illuminated until the disc adjacent to it was depressed. This simultaneously turned off the LED and randomly illuminated another one. The computer recorded the time interval between onset of each LED and depression of the corresponding push button as well as the number of push buttons adjacent to unlit LEDs that were depressed. The subjects used a wooden wand (30 cm long) with a rubber tip (approximately 2 cm × 1.1 cm) to strike the discs. Hypoxia was induced by the inhalation of gas mixtures contained in four high-pressure bottles selected from 11 mixtures that varied in O_2 content from 9.5% to 14.5% O_2 in steps of 0.5%, with the balance nitrogen. Air from another cylinder was breathed as a control. Valves (Matheson, Model 9-580) reduced the pressure from the cylinders to 5.52×10^5 $N \cdot m^{-2}$, and each mixture was then fed into a common valve (Industrial Projects, Model BR5-TSL). This allowed manual selection of any one of the four mixtures to a demand regulator (ARO Model MD-1) that reduced the pressure to ambient level. The breathing mixture was then transmitted to an oral-nasal mask (Canadian Forces, Model A13A)

via flexible corrugated tubing. This arrangement allowed any desired level of SaO_2 to be maintained by monitoring it and switching between breathing mixtures as necessary during the experiment. SaO_2 was measured with an ear oximeter (Hewlett Packard, Model 47201A) and continuously recorded on a strip-chart recorder (Perkin-Elmer Model 023).

Design and Procedure

Six levels of hypoxia were tested, ranging from an SaO_2 of 86% (equivalent to 8900 feet; see Fowler et al., 1985, for details on the conversion of SaO_2 to equivalent altitude) to 76% (11 400 feet) in increments of 2% (500 feet). The upper SaO_2 level was based on pilot studies indicating that SCRT performance was unaffected at or above 86%. Each level was tested in a separate session. For Subjects 1, 2, and 3, the order of testing levels was randomized. For subjects 4, 5, and 6, a descending order from 86% to 76% was used followed by two additional testing sessions at 84% and 82%. Each session consisted of two parts. In each part, response time was assessed three times: first while breathing air, then a hypoxic mixture, and then air again. High-brightness stimuli were presented in one part and low-brightness stimuli in the other, the order of presentation being counterbalanced across sessions.

The task was performed for 90 s by successively striking each push button adjacent to a lighted LED with the wand. Prior to the experiment, the subjects practiced working as quickly as possible while maintaining an error rate of 3 ± 2%. The six sessions were at least one day apart and were approximately 60 min in duration. The first 20 min of each session was devoted to visual acclimatization; then the oral-nasal mask was donned and the six 90-s tests performed in succession. SaO_2 was lowered from the control value (95% to 96%) and stabilized over a 10- to 20-min period using a single blind procedure. Data were discarded and the test repeated immediately if the 3 ± 2% error criterion was exceeded, or if SaO_2 exceeded ± 1% of the appropriate value for 15 s prior to, and during, a test. Maintaining a constant error rate avoided any confounding influence due to a possible shift in the speed/accuracy trade-off setting under hypoxia.

RESULTS

Mean response time for correct first responses (omitting errors and the subsequent error-correcting response) was calculated for each subject in each condition. A 2 (Air Controls) × 6 (Sessions) × 2 (Stimulus Brightness) repeated-measures analysis of variance was performed on these data. Response time was faster to the high than to the low brightness stimuli, $F(1,5) = 26.6$, $p = 0.003$, but no other effects were significant. Therefore, each pair of control values was pooled and the result subtracted from the corresponding hypoxia value to produce a difference score. This latter procedure was adopted to eliminate between-session variations in SCRT performance. The stability of between- and within-session performance can be assessed from Table 1, which shows mean response times for the three subjects who repeated sessions

TABLE 1

Mean Response Time Differences (ms) and (Standard Error of the Means), Pooled across Stimulus Brightness, for Subjects 4, 5, and 6 Who Were Tested Twice at the Same SaO_2 Level

	SaO_2 Level			
Session Number	*84%*		*82%*	
	1	*2*	*1*	*2*
S4 Normoxic	487(11)	472(10)	502(11)	458(9)
Hypoxic	485(12)	476(8)	514(10)	470(9)
S5 Normoxic	418(10)	402(9)	406(10)	384(9)
Hypoxic	416(10)	400(10)	414(10)	394(9)
S6 Normoxic	453(9)	434(10)	448(9)	436(9)
Hypoxic	450(9)	434(10)	456(10)	443(10)

at SaO_2 values of 84% and 82%. These means are based on approximately 225 responses generated during a 90-s run.

The difference scores, excluding the second repetitions, were analysed with a 2 × 6 × 2 three-way analysis of variance with order of testing as a between-subjects factor and SaO_2 level and stimulus brightness as within-subject factors. The main effects of SaO_2 level, $F(5,20) = 81.08$, $p < 0.001$, and stimulus brightness, $F(1,4) = 12.13$, $p < 0.02$, were significant, as was the SaO_2 Level × Stimulus Brightness interaction, $F(5,20) = 2.64$, $p < 0.05$, but no other effects were significant. This analysis indicates that the two different psychometric methods used in the experiment did not influence the results; the mean difference scores were pooled across order of testing and are illustrated in Figure 1. A least-significant-difference post hoc test indicated that, for both the high and low brightness conditions, response time at the SaO_2 levels of 86% and 84% did not differ but was slowed at 82% compared with 84% ($p < 0.01$ in both cases). These results indicate that reliable slowing occurred initially at an SaO_2 level of 82% in both brightness conditions. However, low brightness produced a slight but statistically significant steeper dose-response function than did high brightness.

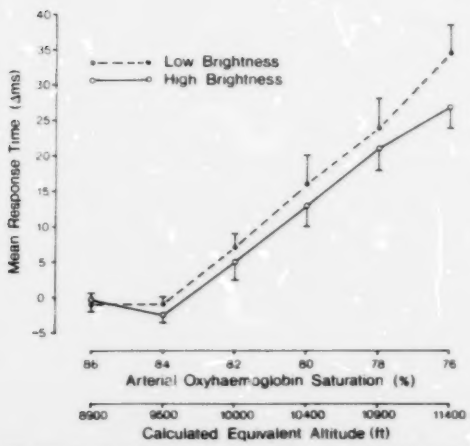

Figure 1. *Response time as a function of SaO_2 (arterial oxyhemoglobin saturation) and stimulus brightness for six subjects. Bars represent standard errors of the mean.*

DISCUSSION

Compared with previous studies, controlling SaO_2 to ±1% (equivalent to an altitude range of approximately 500 feet) resulted in a dose-response function from which a threshold altitude for perceptual-motor decrement due to hypoxia can be estimated with a relatively high degree of accuracy. Slowing of response time was detected at an SaO_2 of 82% but not 84%. Therefore, by interpolation, the best estimate of the threshold is an SaO_2 level of 83% (9750 feet). This is very close to the value of 10 000 feet proposed by Tune (1964) but is well above the estimate of 8000 feet suggested by the results of the Denison et al. (1966) study. Denison et al. (1966) hypothesized that it was the novelty of the Mannikin test that made it particularly sensitive to hypoxia. It might therefore be argued that a somewhat lower threshold would have been found in the present experiment if the SCRT had been novel, since the threshold for the Mannikin test has only been determined to lie between 8000 and 12 000 feet (Fowler et al., 1985). However, there are two problems with this argument. First, when Fowler et al. (1985) eventually demonstrated an effect on the Mannikin test at 12 000 feet, it was not the kind predicted by the task-novelty hypothesis. Second, other experiments have not indicated that learning and memory are particularly sensitive to hypoxia (Crow and Kelman, 1973; Morgan and Green, 1983; Paul, 1983). Of course, the Mannikin test might be more sensitive than the SCRT for reasons other than novelty, but this possi-

bility is little more than speculation on current evidence.

It was also found in this experiment that the slope of the dose-response function was steeper for the low than for the high brightness condition. Thus, reaction time was slowed more rapidly in the former condition as the severity of hypoxia increased. This interaction has also been observed with severe hypoxia by Fowler et al. (1982), who suggested that it could be accounted for by an impairment of brightness discrimination, which is particularly sensitive to hypoxia (Hecht et al., 1946). In this view, impaired brightness discrimination is manifested by a decrease in the apparent brightness of the stimulus, thereby slowing reaction time (Teichner, 1954). Impaired visual functioning has also been suggested as the reason hypoxia increases reaction time to peripherally located flashes of light (Kobrick, 1972; Kobrick and Dusek, 1970). Tichauer (1963) analyzed a complex machine-tool operation at high altitude and found that a critical factor limiting performance was the unreliability of visual feedback compared with either tactile or auditory feedback. These observations suggest that visual impairment can be a relatively important source of variance underlying the hypoxic disruption of perceptual-motor performance.

The threshold estimate of 9750 feet found in this experiment supports the argument by Fowler et al. (1985) that it would be overly conservative to maintain cabin pressure at 8000 feet (Ernsting, 1978, 1984). However, there was some slowing of responding at 10 000 feet. This leaves little margin of safety for an aircrew member breathing cabin air at this altitude. Furthermore, Fowler et al. (1985) have suggested that decreased breathing rate and increased workload can trigger a drop in SaO_2 without variation in cabin pressure, although it is a matter of conjecture whether this combination of factors would ever be likely to occur in an operational environment. In any analysis of this problem, it is useful to keep in mind the distinction between nominal altitude (partial pressure of oxygen in the breathing mixture) and effective altitude (SaO_2 level). The latter appears to be a better and more reliable predictor of performance impairment since the two measures can become uncoupled under certain circumstances. Therefore, effective altitude needs to be assessed independently by monitoring SaO_2 or alveolar gases.

It should also be noted that although the interaction between hypoxia and stimulus brightness was relatively small in this experiment and did not influence the threshold, this might not always be the case. Stimulus brightness in the low condition was well above threshold, and the level of ambient illumination was sufficient for photopic vision to predominate, although scotopic vision is more sensitive to hypoxia (McFarland, 1963). Therefore, under conditions of near-threshold stimuli and a level of illumination requiring scotopic vision, a threshold lower than 9750 feet might be found with the SCRT.

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